# On Early Extinction and the Effect of Travelling in the SIR Model


## Abstract

We consider a population protocol version of the SIR model. In every round, an individual is chosen uniformly at random. If the individual is susceptible, then it becomes infected w.p. $\beta I_t/N$, where $I_t$ is the number of infections at time $t$ and $N$ is the total number of individuals. If the individual is infected, then it recovers w.p. $\gamma$, whereas, if the individual is already recovered, nothing happens.
We prove sharp bounds on the probability of the disease becoming pandemic vs extinguishing early (dying out quickly). The probability of extinguishing early, $\mathbf{Pr}[\mathcal{E}_{ext}]$, is typically neglected in prior work since most use (deterministic) differential equations. Leveraging on this, using $\mathbf{Pr}[\mathcal{E}_{ext}]$, we proceed by bounding the expected size of the population that contracts the disease $\mathbf{E}[R_\infty]$. Prior work only calculated $\mathbf{E}\left[R_\infty \mid \overline{\mathcal{E}_{ext}}\right]$, or obtained non-closed form solutions.
We then study the two-country model also accounting for the role of $\mathbf{Pr}[\mathcal{E}_{ext}]$. We assume that both countries have different infection rates $\beta^{(i)}$, but share the same recovery rate $\gamma$. In this model, each round has two steps: First, an individual is chosen u.a.r. and travels w.p. $p_{travel}$ to the other country. Afterwards, the process continues as before with the respective infection rates.
Finally, using simulations, we characterise the influence of $p_{travel}$ on the total number of infections. Our simulations show that, depending on the $\beta^{(i)}$, increasing $p_{travel}$ can decrease or increase the expected total number of infections $\mathbf{E}[R_\infty]$.


## 1 INTRODUCTION

In this paper we consider the well-known SIR process which is used to study the spread of a contagious disease. The model was introduced in the early 20th century (Ross and Hudson [1917], Kermack and McKendrick [1927]). The population is split into three compartments (or states): susceptible (S), infected (I) and recovered (R). At the beginning of an epidemic, a small number of individuals are infected and the rest of the population is susceptible. Susceptible individuals can be infected and infected individuals can recover and become permanently immune to the disease. The model is often used to study the spread of diseases like COVID-19, measles, mumps and rubella.

When an epidemic starts, the number of infected individuals increases rapidly. At some point, the proportion of still susceptible and already recovered individuals will be such that the spread of the infection will begin to slow down to the point of complete stop. The model is characterised by transition rates between the (compartments/states) depending on the infection rate $\beta$ and the recovery rate $\gamma$.

The so-called reproduction number of an SIR process is defined as $\mathfrak{R}_0 = \beta/\gamma$. This number is equal to the expected number of secondary cases following the introduction of one infected individual into a fully susceptible population. This reproduction number determines how the process evolves over time and how many individuals will get infected until the infection dies out. In general SIR processes behave as follows. If $\mathfrak{R}_0 < 1$, the disease extinguishes early and the number of total infections follows an exponential distribution. If $\mathfrak{R}_0 > 1$, the number of infected individuals first grows exponentially (in expectation). If the number of infected and recovered individuals reaches a fraction of $1 - 1/\mathfrak{R}_0$ the number of infected individuals decreases, in expectation, until it reaches zero. The fraction $1 - 1/\mathfrak{R}_0$ is also called *herd immunity threshold*.

Looking closer at the case $\mathfrak{R}_0 > 1$, one can see that the total number of infections follows a bi-modal distribution. The first peak represents *early extinction*, meaning the process terminates early (see Section 2 for a precise definition). If this unlikely event does not happen, then the process is likely to reach the herd immunity threshold and a much

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larger number of individuals gets infected.

In this paper we study the SIR process as a population protocol. We assume that the population has a fixed size $N$ and that the individuals are modelled as a finite state machine. The state space of our protocol is simple, each individual being in one of the three states $S$, $I$, or $R$. At the beginning of time $t$, we denote by $S_t$, $I_t$ and $R_t$ the number of individuals susceptible to a disease, infected individuals and recovered individuals, respectively. At the beginning of the process $t = 0$, one individual (or a small subset of them) is infected and all other individuals are susceptible. No individual is recovered yet. The process is now defined as follows. In each step a pair of individuals $i, j$ is chosen uniformly at random and is allowed to change state. If the individual $i$ is currently infected, then it recovers with probability $\gamma$. If $i$ is susceptible and $j$ is infected, then $i$ becomes infected with probability $\beta$.

As mentioned before, it may happen that the disease dies out quickly without ever evolving into a pandemic, i.e., without reaching the herd immunity threshold. In this paper we calculate the probability of early extinction as a function of $\mathfrak{R}_0 = \beta/\gamma$ and we present a fully rigorous analysis for such probability using coupling arguments. We show that, if we start with $s$ infected individuals, the probability for early extinction $\mathbf{Pr}\left[\mathcal{E}_{ext}\right]$ is asymptotically $(1/\mathfrak{R}_0)^s$ assuming $\beta > \gamma + \varepsilon$ for some arbitrarily small constant $\varepsilon$. If $\beta < \gamma - \varepsilon$, then $\mathbf{Pr}\left[\mathcal{E}_{ext}\right] = 1 - o(1)$. We then calculate the expected size of the population that gets infected throughout the process $\mathbf{E}\left[R_\infty\right]$. Note that $R_\infty$ denotes the number of recovered individuals at the end of the process. The recovered state is the only terminal state and equals the number of individuals that were infected throughout the process. We first show that $\mathbf{E}\left[R_\infty \mid \overline{\mathcal{E}_{ext}}\right]$ is close to zero. Then we show, conditioning on $\overline{\mathcal{E}_{ext}}$, that $\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right] = \left(1 + W\left(-\mathfrak{R}_0 \cdot e^{-\mathfrak{R}_0}\right)/\mathfrak{R}_0\right) \pm o(1)$, where $r_\infty = R_\infty/N$. Combining our results, we show that, without any conditioning,

$$\mathbf{E}\left[r_\infty\right] = \mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right] \cdot \left(1 - \mathbf{Pr}\left[\mathcal{E}_{ext}\right]\right) \pm o(1).$$

Many of the results found in the literature are based on first-order methods (mean field approaches) (e.g., Kröger and Schlickeiser [2020], Bicher and Popper [2013]) and they give an expression similar to our term for $\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right]$ as an estimation of $\mathbf{E}\left[r_\infty\right]$. This is due to the fact that, with a mean field approach, the process is regarded as deterministic, which means that early extinctions cannot happen for $\mathfrak{R}_0 > 1$. Furthermore, such approach neglects the variance of the process, possibly crucial in the analysis of random processes (see Berenbrink et al. [2017] for a majority type process where the expected change is the same, but the variance in the process determines the convergence time). Although, it has been observed through simulations that the deterministic and stochastic processes differ in terms of the total number of infections (e.g., Fig.13 of Allen and Burgin [2000]).

In the second part of our paper we consider a two-country setting: we show through simulations how travelling affects the spread of the disease. A possible ban, or restriction, on travelling has been considered and applied by governments in some cases, especially with respect to the current pandemic. Such measure falls under the label of non-pharmaceutical interventions (NPI). We introduce an extension of the model where each individual resides in one of two countries. We assume that both countries can have different infection rates, $\beta^{(1)} \neq \beta^{(2)}$, and each country has population size $N^{(1)},\ N^{(2)}$ with $N = N^{(1)} + N^{(2)}$. The process is similar to our original process. First, an individual $i$ is chosen uniformly at random among the whole population of both countries (with probability $1/N$). The chosen individual then travels to the other country with a probability of $p_{travel}$ and remains in the current country w.p. $1 - p_{travel}$. After this, one of the two countries is chosen with a probability proportional to its population and one step of the single-country process is performed in that country. We study, via simulations, the connection between $p_{travel}$ and the total number of infections over time. We obtain interesting and surprising insights. For example, one would think that increasing $p_{travel}$ increases the total number of infections. While this is generally true for small values of $p_{travel}$, (though for some values of $\beta$ and $\gamma$, not even this is true) it turns out that this does not hold if one country has $\mathfrak{R}_0^{(1)} = \beta^{(1)}/\gamma > 1$ and the other country has sufficiently small $\mathfrak{R}_0^{(2)} < 1$. In that case for large enough values of $p_{travel}$, the total number of infections is decreased. Our simulations suggest that early extinction affects the size of the epidemic, leading its average to be reduced and its variance to increase.

### 1.1 RELATED WORK

The SIR model is a very basic mathematical model for the spread of diseases (e.g. it does not model birth and death of individuals, the latter resulting from the illness or other reasons) and a very large number of generalizations were suggested in the literature. Due to the vast literature, we will only discuss results in the SIR comparable to our results and we will concentrate on analytical results. There are also many compartmental models similar to the SIR model. See Li [2018], Daley [1999] for a nice overview about different mathematical models.

The SIR model was introduced in 1927 by Kermack and McKendrick [1927], building on work by Ross and Hudson [1917]. The authors also introduced the well-known set of non-linear ordinary differential equations (which we introduce in this paper too) to describe the spread of the disease. The authors showed the so-called threshold theorem, which predicts the critical fraction of susceptible individuals in the population that must be exceeded if an epidemic is to occur. The authors show that $s_\infty = 1 - r_\infty =$

$s_0 e^{-\mathfrak{R}_0(r_\infty - r_0)}$, where $s_\infty = |S_\infty|/N$ (fraction of susceptible individuals as time goes to infinity), $r_\infty = |R_\infty|/N$, $s_t = |S_t|/N$, and $r_t = |R_t|/N$. This transcendental equation has a solution in terms of the Lambert $W$ function: $s_\infty = -\mathfrak{R}_0^{-1} W(-s_0 \mathfrak{R}_0 e^{-\mathfrak{R}_0(1-r_0)})$.

Most of the publications investigating the SIR model use numerical methods, employing a wide number of different techniques (see Biazar [2006], Rafei et al. [2007a,b] and the references therein). There is a huge amount of literature studying very diverse effects in the SIR compartmental model. Much of it approximates the random process with a deterministic process defined via ordinary differential equations. Hence an early extinction of the process is not possible. In Harko et al. [2014] the authors derived an exact analytical solution to the SIR model in parametric form. A similar result was shown by Miller [2012, 2017]. In Shabbir et al. [2011] an exact analytical solution to the SIR and SIS models with constant population is obtained with the help of direct integration tools. Lefèvre and Simon [2020] propose a block-structured Markov process to describe the spread of epidemics of SIR type and they determine the distribution of the final state of the process. In Black and Ross [2015] the authors present a new method for the recursive computation of the epidemic size distributions. The authors do not estimate the expected size of the epidemic nor do they give a closed form of its distribution.

Pastor-Satorras et al. [2015] give a great overview about epidemic processes in networks: Here the individuals are connected by a network modelling social contacts, such that the infection spreads from a node to its neighbours. The authors of Janson et al. [2014] consider random graphs with a given degree sequence and prove that there is a threshold as a function for $\gamma$, $\beta$, and given vertex degrees. Below the threshold, only a small number of infections occurs, while above it most of the graph gets infected. Kempe et al. [2003] consider influence maximisation in the independent cascade model introduced in Goldenberg et al. [2001a,b]. The process works in parallel rounds and it starts with a set of active (infected) nodes. Every active node infects every non-infected neighbour with a probability of $p$. Then the active nodes become inactive and the newly infected neighbours become active. The process runs until no more activations are possible. The optimization problem of selecting the most influential nodes to be activated in the beginning is NP-hard and the authors provide the first provable approximation guarantees for efficient algorithms. For an overview about results in this model see Shakarian et al. [2015].

Bohman and Picollelli [2012] consider an SIR process of random graphs with a given degree sequence in an continuous time model. Each infected node sends infections to each of its neighbors at times determined by independent exponential random variables with parameter $\lambda$. An infected node recovers at a random time given by an independent exponential random variable with parameter $r_\infty$. The authors assume that the infection spreads from a single infected node and show that either the disease halts after infecting only a small number of nodes, or an epidemic spreads to infect a linear number of nodes. The authors also show that, conditioned on the event that more than a small number of nodes are infected, the epidemic is likely to follow a trajectory given by the solution of an associated system of ordinary differential equations. Their approach gives bounds on the total number of infected nodes.

There is also related work that considers an agent-based modelling of the SIR model (e.g. Bicher and Popper [2013]). These investigate some form of non-homogeneous populations (e.g. distances between agents given by a graph), and analyse results empirically, either using geographic information systems Perez and Dragicevic [2009], or social relationships Alzu'bi et al. [2021].

Many works model how different societal factors play a role in the evolution of an epidemic. In the case of the COVID-19 pandemic, many of them focus on accounting for different factors, like social distancing measures and testing regimens in order to build a reliable model. In Levesque et al. [2021] the authors create a Crump-Mode-Jagers continuous time branching process modelling COVID-19 propagation in order to decide which mitigation strategies are more effective. Similar works developed mathematical and data-driven models in order to establish the efficacy of such measures Sun et al. [2020], Choi and Shim [2021], Liu et al. [2021]. However, there is still a lack of work studying the role travelling has on the epidemic. In Arino and Van Den Driessche [2003], the authors incorporate the concept of travel into the ordinary differential equations for a Susceptible-Exposed-Infected-Removed-Susceptible (SEIRS) model. They derive bounds on $\mathfrak{R}_0$ and show that a disease-free equilibrium is reached, though they hint at its uniqueness through simulations and no discussion is made about the final expected size of the population. Their model is complex and detailed, with the central assumption that individuals come back to the origin country before leaving again for any other country. This is different from our work, where we do not require individuals to follow travel patterns. In Zakary et al. [2017] a multi-regions discrete-time model describes the spatial-temporal spread of an epidemic. Starting from one region, this enters to regions connected with their neighbors by any mean favouring movement. Like in our case, the authors consider homogeneous SIR populations.

## 2 MODELS AND RESULTS

In this section we introduce formally the two models considered in this paper. Both models work in sequential rounds and are population protocols Angluin et al. [2006]. At every round, two individuals $i$ and $j$ are picked uniformly at random. Individual $i$ can either become infected, recover, or stay susceptible, depending on the current state of $i$ and $j$. In

our or second model, individuals can also travel. We study discrete-time models, where in each integer step, an action occurs. One major advantage the discrete-time models have over continuous time models is that they allow to study the early extinction phenomenon. Note that continuous systems and, in particular, differential equations fail to capture early extinctions.

**Single Country Model.** Let $S_t, I_t, R_t$ be the number of susceptible, infected and recovered individuals at time $t$, with $S_t + I_t + R_t = N$, where $N$ is the total number of individuals. Our SIR process is defined as follows. In every round two individuals $i, j$ are picked uniformly at random.

1. If individual $i$ is infected, then it recovers w.p. $\gamma$.
2. If individual $i$ is susceptible and $j$ is infected, then $i$ will becomes infected w.p. $\beta$.

Note that $\beta \cdot I_t/N$ is the probability that the selected susceptible individual $i$ becomes infected. We assume that $\beta$ and $\gamma$ are both constants. In expectation, the system evolves as follows.

$$\begin{aligned}
\mathbf{E}\left[I_{t+1}\right] &= I_t + \beta \cdot (I_t/N) \cdot (S_t/N) - \gamma \cdot (I_t/N) \\
\mathbf{E}\left[S_{t+1}\right] &= S_t - \beta \cdot (I_t/N) \cdot (S_t/N) \\
\mathbf{E}\left[R_{t+1}\right] &= R_t + \gamma \cdot I_t/N
\end{aligned}$$

The reproduction number is defined as $\mathfrak{R}_0 = \beta/\gamma$. This number is equal to the expected number of infections caused by an infected individual assuming that $S = N$. Over time, the number $S_t$ decreases such that every newly infected individual introduces less and less infections into the population. The herd immunity threshold is defined as $1 - 1/\mathfrak{R}_0$. This is the value of $S_t/N$ such that $\mathfrak{R}_0 \cdot S_t/N = 1$.

In Last [2001], a pandemic is defined as "an epidemic occurring worldwide, or over a very wide area, crossing international boundaries and usually affecting a large number of individuals". In this work we assume a simplified notion of pandemic, depending only on the total number of infected individuals. We say a process results in a *pandemic* if at one point of time the number of active infections ($I_t$) reaches $\sqrt{N}$.[1] Moreover, if the process does not result in a pandemic, then we say it *extincts early*. We denote the corresponding events by $\mathcal{E}_{pan}$ and $\mathcal{E}_{ext}$. Finally, we define $R_\infty$ as the *epidemic* final size; since all individuals eventually recover this corresponds to $R_\infty = \lim_{t\to\infty} R_t$. We define $r_\infty = R_\infty/N$. Let $W$ denote the Lambert W-function, which is the inverse of function $f(W) = We^W$ considered for $W \in [-1, \infty)$ (where function $f$ increases monotonically from $-1/e$ to infinity).

[1] Our results suggest that any value $> \log N$ scaled with $\beta$ and $\gamma$ will work, as reaching it will ensure that eventually a constant fraction of $N$ will become infected with very high probability. The reason for $\sqrt{N}$ is it avoids using terms of $\gamma$ and $\beta$ in the definition, assuming they are both constants (independent of $N$).

**Model for two countries.** In this case we assume that the $N$ individuals are distributed over two countries. $N_t^{(1)}$ and $N_t^{(2)}$ denotes the number of individuals staying in country 1 and 2 at time $t$. We assume $N_t^{(1)} = N_t^{(1)} = N/2$. The number of susceptible, infected and recovered individuals at time $t$ in country $i$ is denoted by $S_t^{(i)}, I_t^{(i)}$ and $R_t^{(1i)}$ for $i \in \{1, 2\}$. Initially we have $I_t^{(1)} = I_t^{(2)} = s$ for $s \geq 1$. We assume that every country has its own infection rate: $\beta^{(1)}$ and $\beta^{(2)}$. The recovery rate in both countries is $\gamma$. The process has two selection steps and step $t$ works as follows.

1. Pick an individual $i$ uniformly at random (with prob. $1/N$). With probability $p_{travel}$ individual $i$ travels from its current country to the other country. Adjust $N_t^{(1)}$ and $N_t^{(2)}$ accordingly.
2. Pick country $\ell$ as follows: $\ell = 1$ w.p. $N_t^{(1)}/N$ and 2 otherwise.

   An SIR step as described above is applied on the chosen country.

   (a) Pick a pair of individuals $i$ and $j$ uniformly at random from country $\ell$.
   (b) If individual $i$ is infected, then it recovers w.p. $\gamma$.
   (c) If individual $i$ is susceptible and $j$ is infected, then $i$ will become infected w.p. $\beta^{(\ell)}$.

Compared to existing works, this model is simpler and posing minimal constraints to the movement of individuals. This makes it easily adaptable, for example in the case of a change in the number of countries involved. Further, a simpler model might facilitate future analytical results regarding the expected size of the epidemic.

## 2.1 RESULTS

Our main result Theorem 4 shows a very tight characterisation of the early extinction probability. Recall that a process extincts early if the number of infected dies out before ever reaching $I_t \geq \sqrt{N}$, where $N$ is the size of the population. Recall that $\mathfrak{R}_0 = \beta/\gamma$ is the reproduction number, $R_\infty$ is the total number of infected individuals and $r_\infty = R_\infty/N$ . First we present a simplified version of Theorem 4.

**Theorem 1** (Simplified version of Theorem 4)**.** *Let $\beta + \gamma \leq 1$. Consider the single country model starting with $I_0 = s \leq \frac{\sqrt{N}}{2 \log N \log\log N}$ infections. Let $\varepsilon = 5 \log N/\sqrt{N}$ and let $\mathbf{Pr}\left[\mathcal{E}_{ext}\right]$ be the probability of early extinction.*

1. *If $\beta < \gamma - \varepsilon$, then $\mathbf{Pr}\left[\mathcal{E}_{ext}\right] \geq 1 - o(1)$.*
2. *If $\beta > \gamma + \varepsilon$, then $\mathbf{Pr}\left[\mathcal{E}_{ext}\right] = (1/\mathfrak{R}_0)^s \pm o(1)$.*

For the case where $\beta > \gamma$ and $s = 1$ the probability of early extinction is essentially $1/\mathfrak{R}_0 = \gamma/\beta$. If $s > 1$ the probability decreases exponentially in $s$. If $\beta < \gamma$, then the process is very likely to reach an early extinction. We

leave the case $\beta = \gamma$ open but note that $\varepsilon$ will be arbitrary small with growing $N$. The challenging part in the proof of Theorem 4 is non-linear update rule of our process. Note that rewriting (1)-(1) by replacing $R_t = N - S_t - I_t$ reveals the non-linearity. To overcome these challenges, we use a series of couplings, allowing which allows us to relate our SIR process to a biased random walk.

Note that in the above result $\mathcal{E}_{ext}$ is defined as the event of having $\sqrt{N}$ many infected individuals at the same time. Hence, it is still possible that $R_\infty = \Omega(N)$, i.e., at the end of the process the number of recovered individuals (which equals the total number of infections) is linear in $N$. In Proposition 1 (see Section 3.1) we show that this is not the case; in the event of $\mathcal{E}_{ext}$ the number of recovered individuals never exceeds $\sqrt{N}\log N$, w.h.p. This means that our bounds on the the early extinction still hold even if the definition was changed to requiring $R_\infty \leq \sqrt{N}\log N$.

The next theorem shows a bound on the the expected number of total infections $\mathbf{E}[R_\infty]$, expressed in terms of the Lambert function $W$. Note that $W(x) < 0$ for $x \in [-1/e, 0)$. As mentioned before, many approaches like first-order methods, mean field approaches and ordinary differential equations ODE (see e.g., Kröger and Schlickeiser [2020], Bicher and Popper [2013]), cannot account for early extinction due to the underlying determinism. Instead, they obtain bounds of the kind $\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right]$. There has also been been some work based on stochastic differential equations (e.g., Allen [2008], Williams et al. [2012]), complemented with simulations results. We are not aware of results based on SDEs that obtain closed-form results for $\mathbf{E}[r_\infty]$.

**Theorem 2.** *Assume that $|\beta - \gamma| \geq \varepsilon$ for an arbitrarily small constant $\varepsilon$. Assume $N = N(\varepsilon)$ is large enough. Let $W : [-1/e, \infty) \longrightarrow [-1, \infty)$ be the Lambert function. Consider the single country model starting with $I_0 = s \leq \frac{\sqrt{N}}{2\log N \log\log N}$ infections.*

1. *If $\beta < \gamma - \varepsilon$, then the expected total number of infections is sublinear, i.e., $\mathbf{E}[r_\infty] = o(1)$.*
2. *If $\beta > \gamma + \varepsilon$, then $\mathbf{E}[r_\infty] \sim \left(1 + \frac{W\left(-\mathfrak{R}_0 \cdot e^{-\mathfrak{R}_0}\right)}{\mathfrak{R}_0}\right)\left(1 - \left(\frac{1}{\mathfrak{R}_0}\right)^s\right)$.*

*See Fig. 4 in the Appendix for a depiction.*

The following theorem estimates the expected number of infections conditioned on not having early extinction. It is used to show Theorem 2. The following theorem yields a result similar to the one obtained through first-order methods used by Allen [2008], Williams et al. [2012], but adapted to our population based model. In Theorem 6 (in Section 4) we provide a generalisation of the following theorem.

**Theorem 3.** *Consider the single country model. Assume that $\beta > \gamma$ are constants. Let $W : [-1/e, \infty) \longrightarrow [-1, \infty)$ be the Lambert function. Then,*

$$\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right] \sim \left(1 + \frac{W\left(-\mathfrak{R}_0 \cdot e^{-\mathfrak{R}_0}\right)}{\mathfrak{R}_0}\right). \quad (1)$$

*Furthermore, $r_\infty$ is concentrated around its expected value.*

# 3 EARLY EXTINCTION

The SIR model described in Section 2, often results in herd-immunity, where a large fraction of the population was infected such that only very few susceptible individuals remain. From then on, the number of infections decreases slowly until it finally reaches zero. However, in some case, even when $\beta > \gamma$, it can happen that the number of infections remains low and the virus vanishes very suddenly. This is what we refer to as an *early extinction* and which is the focal point of this section. We will derive bounds on the probability of an early extinction in terms of the parameters $\gamma$ and $\beta$. The formal statement is given in the next theorem and implies the theorem given in Theorem 1.

**Theorem 4.** *Consider the single country SIR model as described in Section 2. Define $\tau = \sqrt{N}\log N$, $\varepsilon = 5\log N/\sqrt{N}$, and $\varepsilon' = \varepsilon(N-\tau)/N$, and assume $I_0 = s$ for any $s \leq \frac{\sqrt{N}}{2\log N \log\log N}$. Then we have for $\beta > \gamma + \varepsilon$*

$$\left(\frac{\gamma}{\beta}\right)^s - \frac{3}{N} \;\leq\; \mathbf{Pr}\left[\mathcal{E}_{ext}\right] \;\leq \left(1 + \frac{s\tau}{N - s\tau}\right)\left(\frac{\gamma}{\beta}\right)^s + \frac{2}{N}$$

*and for $\beta < \gamma - \varepsilon$*

$$1 - \left(\frac{\gamma}{\beta}\right)^{s-\sqrt{N}} - \frac{2}{N} \;\leq\; \mathbf{Pr}\left[\mathcal{E}_{ext}\right] \;\leq\; 1$$

To show Theorem 4 our approach is as follows. Ideally, we would like to bound the number of infections as a (biased) random walk over the number of infected individuals. The probability that such a random walk reaches either of its end points can be limited by a bound like the one given in Theorem 7. Unfortunately, here the probability to 'walk' from $I_t$ to $I_t - 1$ or $I_t + 1$ is a function of both $I_t$ and $S_t$. We avoid the dependency on $I_t$ by considering *active* steps only. Recall, these are steps in which the number of infected individuals either increases by one or decreases by one. This allows us to drop the terms $I_t/N$ in the transition probabilities. We circumvent the dependency on $S_t$ by defining a family of processes and by coupling them which each other (see Section 3.1). The processes will be defined and motivated later in this section. Once both problems are solved, we are left with four biased random walks (upper vs lower bounds and $\beta < \gamma$ vs $\beta > \gamma$) that we analyse using Theorem 7 (see Appendix A). This allows us in Theorem 4 to derive almost tight bounds on $\mathbf{Pr}\left[\mathcal{E}_{ext}\right]$.

## 3.1 COUPLINGS

In this section our goal is to show Theorem 5 which upper and lower bounds the probability of early extinction of our

process by the extinction probability of two biased random walk process. Theorem 5 shows that the approximation error is tiny and has only a second-order impact on the extinction probability. In order to prove Theorem 5, we will define two intermediate processes and provide three pairwise couplings.

As a first step, in the entire section, unless stated otherwise, we only consider active steps, i.e., steps where $I_{t+1} \neq I_t$. The subscript $t$ counts now only the active steps. Before defining the processes we give some definitions. Recall that $\mathcal{E}_{ext}$ (early extinction) is the event that the number of infections reaches zero before ever having $\sqrt{N}$ infected individuals at the same time.

Define stopping time as $T = \min_t \left\{ I_t = 0 \text{ or } I_t = \sqrt{N} \right\}$. Hence, $T$ refers to the number of active steps until there are either zero or $\sqrt{N}$ many infected individuals, and $\mathcal{E}_{ext}$ is the event that $I_T = 0$. For any SIR process $P$, we denote by $\xi_t^{(P)}$ the configuration of $P$ at time $t$, which is given by the triplet $\xi_t^{(P)} = \left( S_t^{(P)}, I_t^{(P)}, R_t^{(P)} \right)$. We define $\tau = \sqrt{N} \log N$ and a second stopping time $T_\tau = \min_t \{ I_t = 0, I_t = \sqrt{N} \text{ or } t = \tau \} = \min\{T, \tau\}$.

Compared to the stopping time $T$, here the process does not only stop if the number of infected individuals reaches zero or $\sqrt{N}$ but also if that does not happen during the first $\tau$ (active) time steps. All our processes are stopped at time either $T$ or $T_\tau$. We assume that for every $t$ larger that the stopping time we have $\xi_t^{(P)} = \xi_T^{(P)}$ or $\xi_t^{(P)} = \xi_{T_\tau}^{(P)}$, respectively. Now we are ready to define our processes, which we call $P, P_{\tau,*}, P_{*,S}, P_{\tau,S}$.

1. $P$ is our original process with the stopping time $T$.
2. $P_{\tau,*}$ is our original process with the stopping time $T_\tau$.
3. For $S \in \{N - \tau, N\}$, we define the following biased random walk process $P_{*,S}$.
   At each round the following is done.
   (a) Draw random numbers $X_I, X_S, X_a$ i.i.d. and u.a.r. from $[0, 1]$
   (b) If $X_I \in [0, I_t^{(P_{*,S})}/N)$, then
      i. if $X_a \in [0, \beta)$ and $X_s \in [0, S/N]$, increase $I_t^{(P_{*,S})}$;
      ii. if $X_a \in [\beta, \beta + \gamma)$, decrease $I_t^{(P_{*,S})}$.

   The nice property about process $P_{*,S}$ is that (since we only consider active time steps) the process behaves exactly like a biased random walk. The increase probability of $I_t$ is $\beta S/N$ and the decreas probability is $\gamma$. For this process we apply the stopping time $T$.
4. For $S \in \{N - \tau, N\}$, we define the process $P_{\tau,S}$. $P_{\tau,S}$ behaves like $P_{*,S}$ but we apply the stopping time $T_\tau$.

Table 1 sums up the four processes, while Fig. 1 shows the couplings we are going to prove in the following sections. In this section we will show the following result.

| | no time limit | time limit $\tau$ |
|---|---|---|
| $S$ not fixed | $P$ | $P_{\tau,*}$ |
| $S$ fixed | $P_{*,S}$ | $P_{\tau,S}$ |

Table 1: Description of the processes.

$$P \overset{\text{Prop 1}}{\longleftrightarrow} P_{\tau,*} \overset{\text{Prop 2}}{\longleftrightarrow} P_{*,S} \overset{\text{Prop 3}}{\longleftrightarrow} P_{\tau,S}$$

Figure 1: Diagram of the couplings

**Theorem 5.** *Consider the processes $P$ and $P_{*,S}$. We have*

$$\mathbf{Pr}\left[I_T^{(P_{*,N-\tau})} = \sqrt{N}\right] - 2/N \leq \mathbf{Pr}\left[I_T^{(P)} = \sqrt{N}\right] \leq$$
$$\leq \mathbf{Pr}\left[I_T^{(P_{*,N})} = \sqrt{N}\right] + 2/N, \quad \textit{and}$$
$$\mathbf{Pr}\left[I_T^{(P_{*,N})} = 0\right] - 2/N \leq$$
$$\mathbf{Pr}\left[I_T^{(P)} = 0\right] \leq \mathbf{Pr}\left[I_T^{(P_{*,N-\tau})} = 0\right] + 2/N$$

From this it follows that we can analyse the early extinction time of $P_{*,S}$ instead of $P$. To show Theorem 5 we use 3 couplings: Proposition 1 couples processes $P$ and $P_{\tau,*}$, Proposition 2 couples $P_{\tau,*}$ and $P_{\tau,S}$, and Proposition 3 connects $P_{\tau,S}$ and $P_{*,S}$.

**Proposition 1.** *With prob $1 - 1/N$ we have $I_T^{(P)} = I_{T_\tau}^{(P_{\tau,*})}$.*

**Proposition 2.** *Consider the processes $P_{\tau,*}$ and $P_{\tau,S}$ for $S \in \{N, N - \tau\}$. Let $P = \mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,*})} = \sqrt{N}\right]$. We have $\mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,N-\tau})} = \sqrt{N}\right] \leq P \leq \mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,N})} = \sqrt{N}\right]$.*

Proofs can be found in Appendix C and Appendix D, resp.

**Proposition 3.** *Consider the processes $P_{\tau,S}$ and $P_{*,S}$ for $S \in \{N - \tau, N\}$. Then we have*

$$\mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,N})} = \sqrt{N}\right] \leq \mathbf{Pr}\left[I_T^{(P_{*,N})} = \sqrt{N}\right] + 1/N, \textit{ and}$$
$$\mathbf{Pr}\left[I_T^{(P_{*,N-\tau})} = \sqrt{N}\right] - 1/N \leq \mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,N-\tau})} = \sqrt{N}\right]$$

*Proof.* This proof is similar to proof of Proposition 1. □

Putting all three couplings together yields Theorem 5. From this we are finally able to conclude Theorem 4, since now we can analyse a biased random walk instead. The proof distinguishes between two cases, depending on whether $\beta$ or $\gamma$ is larger and builds on Theorem 7. It can be found in the supplementary material.

## 4 TOTAL NUMBER OF INFECTIONS

In this section we analyse the total number of infections for the SIR model for one country. The proof of the next result can be found in Appendix B.

**Theorem 6.** *Consider the single country model. Assume that $\beta - \gamma \geq \varepsilon$ for an arbitrarily small constant $\varepsilon$. Assume $N = N(\varepsilon)$ is large enough. Let $W : [-1/e, \infty) \longrightarrow [-1, \infty)$ be the Lambert function. Consider a time step $t^* = \omega(1)$. Let $\mathcal{E}'$ be the event $I_{t^*} \in [\omega(1), o(N)]$ and $R_{t^*} = o(N)$. Then,*

$$\mathbf{E}\left[r_\infty \mid \mathcal{E}'\right] \sim \left(1 + \frac{W\left(-\mathfrak{R}_0 \cdot e^{-\mathfrak{R}_0}\right)}{\mathfrak{R}_0}\right). \qquad (2)$$

*Furthermore, $r_\infty$ is concentrated around its expected value.*

## 5 SIMULATIONS FOR TWO COUNTRIES

In this section, we study the impact of travelling and its relationship to early extinction on the total number of infections. Recall that $p_{travel}$ is defined as the travelling rate. Without loss of generality we set $\gamma = 0,2$ in all our simulations.[2] We set the initial size of each country to be $N_0^{(1)} = N_0^{(2)} = 2000$. Note that these size vary to a small extent throughout the process due to traveling individuals. The total population is $N = N_0^{(1)} + N_0^{(2)} = 4000$. Initially, there is one infection per country ($I_0^{(1)} = I_0^{(2)} = 1$) and the rest of the population is susceptible. In our simulations, we vary the travelling rate $p_{travel}$ and the infection rates $\beta^{(1)}$ and $\beta^{(2)}$. W.l.o.g. we assume $\beta^{(1)} = 0 \leq \beta^{(2)} = 0$. For each value of $p_{travel}$, $\beta^{(1)}$, and $\beta^{(2)}$, we output the average of 10.000 iterations. The outcome of the simulations can be found in Fig. 2 and Fig. 3. For each value of $p_{travel}$ we plot the average number of infections $\widehat{R_\infty^{(1)}}$ in country one in light green and in light blue we plot the average number of infections in country two $\widehat{R_\infty^{(2)}}$. Note that we write $\widehat{R_\infty}$ instead of $\mathbf{E}\left[R_\infty\right]$ to emphasise the difference between the average and the expected value. The total number of infections across both countries $\widehat{R_\infty} = \widehat{R_\infty^{(1)}} + \widehat{R_\infty^{(2)}}$ is plotted in red. We plot the smoothed version of $\widehat{R_\infty}$ in black. Let $R_\infty(p_{travel})$ denote the total number of infections for a given travelling rate $p_{travel}$. Finally, the corresponding standard deviation is plotted using vertical bars.

First, note that the standard deviation appears large: this is unavoidable and due to the fact that early extinctions (in this case, when $\widehat{R_\infty} \leq \sqrt{2N} = \sqrt{4000} \approx 63$) occur with constant probability. Excluding runs with an early extinction, the standard deviation becomes negligible. Another effect of this exclusion is that the values of $\widehat{R_\infty}$ turn up to be visibly smaller (see Fig. 2).

It is perhaps natural to assume that increasing $p_{travel}$ also increases $\widehat{R_\infty}$. While this is true for some values of the $\beta$s, there are also values for which $\widehat{R_\infty}$ first increases monotonically and then decreases monotonically. In addition, for large $p_{travel}$ the number of total infections can drop below the value where no travelling occurs, i.e., $\widehat{R_\infty}(1) < \widehat{R_\infty(0)}$. See Fig. 3a and Fig. 3b. Our simulation results can be found in Table 2, Fig. 2 and Fig. 3.

[2] By changing $\beta$ one can get arbitrary infection rates $\mathfrak{R}_0$. Similarly, one can vary $p_{travel}$ to obtain arbitrary rations of $p_{travel}/\gamma$.

| $\beta^{(1)}, \beta^{(2)}$ \ $p_{travel}$ | 0 | 0.05 | 0.1 | 0.2 | 0.5 | 0.95 |
|---|---|---|---|---|---|---|
| 0, 0.3 | 395 | 293 | 121 | 32 | 12 | 10 |
| 0, 0.5 | 1078 | 1514 | 1491 | 1352 | 1025 | 857 |
| 0, 0.8 | 1470 | 2266 | 2416 | 2497 | 2499 | 2500 |
| 0.2, 0.7 | 1399 | 2489 | 2655 | 2695 | 2679 | 2798 |
| (*) 0.2, 0.7 | 1899 | 3181 | 3373 | 3454 | 3460 | 3445 |
| 0.3, 0.3 | 763 | 1276 | 1301 | 1282 | 1305 | 1294 |
| 0.3, 0.5 | 1467 | 2357 | 2433 | 2464 | 2419 | 2416 |
| 0.3, 0.8 | 1868 | 2926 | 3085 | 3132 | 3189 | 3185 |
| 0.5, 0.8 | 2543 | 3367 | 3407 | 3428 | 3443 | 3465 |
| 0.8, 0.8 | 2953 | 3673 | 3687 | 3688 | 3679 | 3682 |

Table 2: The table shows our simulation results for $\widehat{R_\infty}(p_{travel})$ given different values of $\beta$s over 10.000 iterations, with the exception of $(0.2, 0.7)$ – where early extinction (EE) runs are excluded (*) – that is averaged over 1000 iterations. We round to the nearest integer.

**Simulation Result 1.** *Assume that $\gamma = 0.2$.*

1. *$\beta^{(1)} = 0$ and $\beta^{(2)} = 0.3$ results in $R_\infty(p_{travel})$ being non-monotone. Moreover, $R_\infty(1/2) = O(1)$ (Fig. 3a).*
2. *$\beta^{(1)} = 0$ and $\beta^{(2)} = 0.5$ results in $R_\infty(p_{travel})$ being non-monotone. (Fig. 3b).*
3. *$\beta^{(1)} = 0.5$ and $\beta^{(2)} = 0.8$ results in $R_\infty(p_{travel})$ increasing monotonically. (Table 2, and also Fig. 3c). See further examples in Appendix H*

Based on this, we believe that the simulation results can be generalised to the following conjecture.

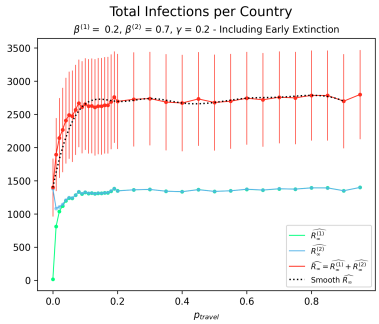
**Conjecture 1.** *For all $\beta^{(1)}, \beta^{(2)}$ and $\gamma$ we have*

*(i)* $\beta^{(1)}, \beta^{(2)} < \gamma$: $\mathbf{E}\left[R_\infty(p_{travel})\right] = O(1)$ *for all* $p_{travel} \in [0, 1]$.

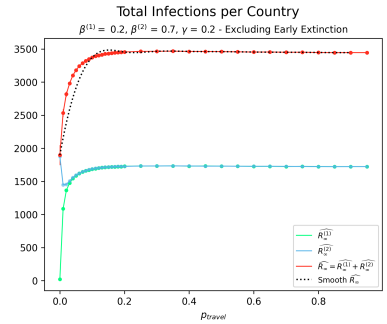
*(ii)* $\beta^{(1)} < \gamma < \beta^{(2)}$ *is non-monotone.*

*(iii)* $\beta^{(1)}, \beta^{(2)} > \gamma$: $\mathbf{E}\left[R_\infty(p_{travel})\right]$ *increases monotonically. Note that Fig. 3c suggests that it is important for the $\beta$s to be strictly larger than $\gamma$ in order for $\mathbf{E}\left[R_\infty(p_{travel})\right]$ to increase monotonically.*

At the core of the conjecture is the following observation. As $p_{travel}$ increases, both $\beta^{(1)}$ and $\beta^{(2)}$ are blended together resulting a linear combination of both values. Consider the following thought experiment where the $\beta$s fully blend until the "effective" $\beta$ of both cities is the average $\overline{\beta} = (\beta^{(1)} + \beta^{(2)})/2$. If we ignore the effect of travelling all statements of the conjecture follow from the Theorem 2. We see in Fig. 3a that if $\overline{\beta}$ is below $\gamma$, then the total number of infections $R_\infty$ is close to 0. For $\overline{\beta}$ larger than $\gamma$, $R_\infty$ is

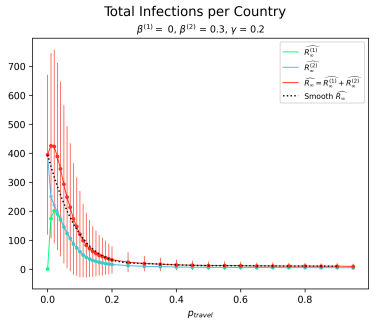



(a) Including early extinction.

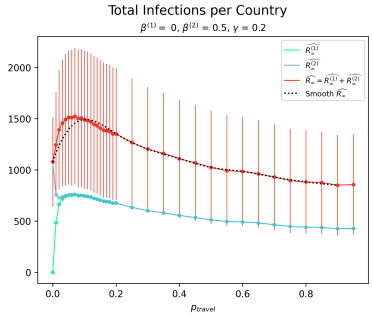



(b) Excluding early extinction.

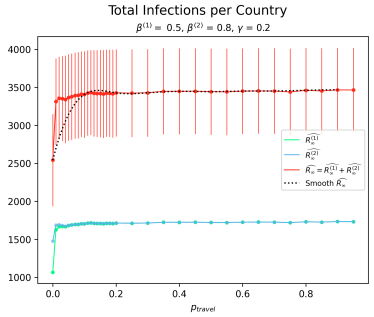
Figure 2: The plots show $r_\infty$ as function of $p_{travel}$ averaged across 1000 iterations. We consider two different settings. In the plot of the l.h.s. we plot $\widehat{R_\infty}$. For the plot on the r.h.s. we plot $\widehat{R_\infty}$ conditioned on $\overline{\mathcal{E}_{ext}}$. The plots shows that $\widehat{R_\infty}$ conditioned on $\overline{\mathcal{E}_{ext}}$ has only very little standard deviation whereas the standard deviation of $\widehat{R_\infty}$ is considerably larger. However, this is unavoidable since there is a positive probability of an early extinction in which case the total infections is close to 0. Therefore, the standard deviation is naturally of linear size. Note that in all other experiments we have ten times more iterations suggesting an adequate number of iterations in our experiments.




(a) $\beta^{(1)} < \gamma < \beta^{(2)}$ and $\overline{\beta} < \gamma$. The total number of infections $\widehat{R_\infty}(p_{travel})$ appears to be non-monotone.




(b) $\beta^{(1)} < \gamma < \beta^{(2)}$ and $\overline{\beta} > \gamma$. The total number of infections $\widehat{R_\infty}(p_{travel})$ appears to be non-monotone.




(c) $\beta^{(1)}, \beta^{(2)} > \gamma$. The total number of infections $\widehat{R_\infty}(p_{travel})$ appears to be monotonically increasing. See also Table 2.

Figure 3: Simulation results for $\widehat{R_\infty}$ varying $p_{travel}$.

concave. The concavity is the key to the understanding. For example, when both original $\beta$s were above $\gamma$, then having $\overline{\beta}$ in both countries increases $R_\infty$, due to the concavity. Let us consider the cases of the conjecture one by one. Part (i) follows since $\overline{\beta} < \gamma$ and any blending of the $\beta$s will yield "effective" $\beta$s less than $\gamma$. For part (ii) note that when $p_{travel} = 0$ and no blending of the $\beta$s occurs, then we have that in one city $\mathfrak{R}_0 > 1$ (expected pandemic) and in the other $\mathfrak{R}_0 < 1$ (expected early extinction). When $p_{travel}$ is slightly above 0 the the blending of the $\beta$s effectively decreases $\beta^{(2)}$ a little bit, the number of infectable individuals also doubles once $p_{travel} > 0$. This becomes clear when one considers the setting where $\beta^{(1)} = 0$ and $\beta^{(2)} = 1$. Here $\mathbf{E}[R_\infty] > N$ and thus clearly some individuals of country 1 contribute. Part (iii) follows immediately from the concavity.

The simulations presented here are only a small fraction of the number of our simulations covering the large variety of different values for $\beta^{(1)}, \beta^{(2)}$ and $\gamma$. All our simulations confirmed our conjecture. Nonetheless, we were not able to turn the arguments above into a rigorous proof. On one hand, considering one country only is already immensely complicated and a second country increases the number of variables and their decencies even further. On the other hand, $\mathbf{Pr}[\mathcal{E}_{ext}]$ and further considerations have to be accounted for. Indeed, one cannot simply ignore $p_{travel}$ and assume that both countries have $\overline{\beta}$. The simulations suggest that we have essentially two values for $R_\infty$; one being $R_\infty(0)$ and one being $R_\infty(p)$, for any $p > 0$ ( See Appendix H). The reason for this is that if $p_{travel} = 0$, then it can happen that one city has an early extinction. When $p_{travel} > 0$, we believe that whenever only one of the countries has an early extinction, then the other country will eventually send infected individuals over and rekindle the infection until it succeeds. While the above shows the influence of effects when $p_{travel}$ is small, when believe that for large values of $p_{travel}$ our arguments capture the behaviour of the process.

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# A APPENDIX: PROOF OF THEOREM 4 - PROBABILITY OF EARLY EXTINCTION

In this section we prove Theorem 4 using the coupling result from Theorem 5 which allow us bound the probability of the the early extinction time of our process by $\mathbf{Pr}\left[\mathcal{E}_{ext}^{(P_{*,S})}\right]$, the early extinction time of $P_{*,S}$ for $S = N$ and $S = N-\tau$. The nice property about $P_{*,S}$ is that, restricted to active time steps, the process behaves exactly like a biased random walk. In the following we will omit the superscript $P_{*,S}$ and write $I_t, S_t$ and $R_t$ for the infected, susceptible and recovered individuals. When clear from the context, we will omit the index $t$. Hence, the probability to increase $I_t$ by 1 is $\beta S/N$ and the probability to decrease $I_t$ is $\gamma$. In more detail

$$\mathbf{Pr}\left[I_{t+1} = I_t + 1\right] = \frac{\beta \frac{S}{N}\frac{I_t}{N}}{\gamma \frac{I_t}{N} + \beta \frac{S}{N}\frac{I_t}{N}} = \frac{\beta S}{\gamma N + \beta S} =: p \tag{3}$$

With this is mind, we consider the ratio $\frac{1-p}{p}$, which is equal to

$$\frac{1-p}{p} = \frac{\gamma N/(\gamma N + \beta S)}{\beta S/(\gamma N + \beta S)} = \frac{\gamma N}{\beta S}.$$

We can apply Theorem 7 in Appendix G, and derive

$$\mathbf{Pr}\left[\mathcal{E}_{ext}\right] = \mathbf{Pr}\left[I_T = 0\right] = \frac{\left(\frac{1-p}{p}\right)^{\sqrt{N}} - \left(\frac{1-p}{p}\right)^{s}}{\left(\frac{1-p}{p}\right)^{\sqrt{N}} - 1} \tag{4}$$

where $I_0 = s$. We separate the proof in two cases, depending on whether the quantity $\left(\frac{\gamma N}{\beta S}\right)$ is smaller or bigger than 1. These cases are, respectively: $\beta > \gamma + \varepsilon'$ and $\beta < \gamma - \varepsilon$. We define $\varepsilon' = \frac{\varepsilon(N-\tau)}{N}$, as reported in the theorem statement.

**Case 1:** $\beta > \gamma + \varepsilon'$**.** From Theorem 5 we have

$$\frac{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - \left(\frac{\gamma}{\beta}\right)^{s}}{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - 1} - \frac{2}{N} \le \mathbf{Pr}\left[\mathcal{E}_{ext}\right] \le \frac{\left(\frac{\gamma N}{\beta(N-\tau)}\right)^{\sqrt{N}} - \left(\frac{\gamma N}{\beta(N-\tau)}\right)^{s}}{\left(\frac{\gamma N}{\beta(N-\tau)}\right)^{\sqrt{N}} - 1} + \frac{2}{N} \tag{5}$$

The left-hand side of Eq. (5) can be lower-bounded as

$$\mathbf{Pr}\left[\mathcal{E}_{ext}\right] \ge \frac{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - \left(\frac{\gamma}{\beta}\right)^{s}}{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - 1} - \frac{2}{N} \ge \left(\frac{\gamma}{\beta}\right)^{s} - \left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - \frac{2}{N} \tag{6}$$

while the right-hand side as

$$\mathbf{Pr}\left[\mathcal{E}_{ext}\right] \le \frac{\left(\frac{\gamma N}{\beta(N-\tau)}\right)^{\sqrt{N}} - \left(\frac{\gamma N}{\beta(N-\tau)}\right)^{s}}{\left(\frac{\gamma N}{\beta(N-\tau)}\right)^{\sqrt{N}} - 1} + \frac{2}{N} \le \left(\frac{\gamma N}{\beta(N-\tau)}\right)^{s} + \frac{2}{N} \tag{7}$$

We get that

$$\mathbf{Pr}\left[\mathcal{E}_{ext}\right] \le \left(\frac{\gamma N}{\beta \cdot (N-\tau)}\right)^{s} + \frac{2}{N} \le \left(\frac{\gamma}{\beta}\right)^{s}(1+x)^{s} + \frac{2}{N}$$

for $x = \tau/(N-\tau)$. We have $(1+x)^s \le 1 + \frac{sx}{1-sx+x}$ for all $x \in [-1, 1/(s-1)]$ and $s > 1$. Moreover,

$$x = \tau/(N-\tau) < 2\log N/\sqrt{N} \le 1/s \le 1/(s-1).$$

Using $x = \tau/(N-\tau)$ we get

$$\begin{aligned}(1+x)^s &\le 1 + \frac{s\tau/(N-\tau)}{1 - s\tau/(N-\tau) + \tau/(N-\tau)} \\ &= 1 + \frac{s\tau}{N - s\tau} \\ &= 1 + o(1)\end{aligned}$$

**Case 2:** $\beta < \gamma - \varepsilon$**.** The left-hand side of Eq. (5) can be lower-bounded by

$$\mathbf{Pr}\left[\mathcal{E}_{ext}\right] \ge \frac{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - \left(\frac{\gamma}{\beta}\right)^{s}}{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}} - 1} - \frac{2}{N} \ge \tag{8}$$

$$1 - \frac{\left(\frac{\gamma}{\beta}\right)^{s}}{\left(\frac{\gamma}{\beta}\right)^{\sqrt{N}}} - \frac{2}{N} = 1 - \left(\frac{\gamma}{\beta}\right)^{s-\sqrt{N}} - \frac{2}{N} \tag{9}$$

# B APPENDIX: PROOF OF THEOREM 6

Let $Z_t = I_t + R_t = N - S_t$ be the total number of infections after $t$ steps with $Z_0 = I_0 + R_0$. We can write $\mathbf{E}\left[I_{t+1} \mid I_t\right]$ as $\mathbf{E}\left[I_{t+1} \mid I_t\right] = I_t \cdot \left[1 + \frac{\beta}{N} \cdot \frac{S_t}{N} - \frac{\gamma}{N}\right]$. For the analysis we consider active steps only, i. e., steps $t$ in which the number of infected particles either increases by one (new infection: $I_{t+1} = I_t + 1$), or decreases by one (new recovery: $I_{t+1} = I_t - 1$, $R_{t+1} = R_t + 1$). In the first case $S_{t+1} = S_t - 1$ and $Z_{t+1} = Z_t + 1$; in the second case, $S_{t+1} = S_t$ and $Z_{t+1} = Z_t$.

Let $L$ be the number of individual infected during the whole process, not counting the initial $I_0$ infections (i.e., $R_\infty = I_0 + R_0 + L$), and for $j = 0, 1, \ldots, L$, let $T_j \ge 0$ be the length (in active steps) of the $j$-th recovery run. That is, $T_j$

is the number of recoveries between the $j$-th and $(j+1)$-st infections (or, for $j = L$, the number of the remaining recoveries after the very last infection in the process). Let $T(j) = T_0 + T_1 + \cdots + T_j$. For $j = 0, 1, \ldots, L$, after the $j$-th recovery run, that is, after $T(j) + j$ active steps (which are $T(j)$ recoveries plus one infection after each recovery run except the last one), the parameters $S$, $I$ and $R$ are $S^{(j)} = S_0 - j$, $I^{(j)} = I_0 + j - T(j)$, $R^{(j)} = R_0 + T(j)$.

After the last recovery run, $I^{(L)} = 0$ (no infected individuals left), so $I_0 + R_0 + L - T(L) = 0$, and we want to use this equation to derive $L$ (and hence also $R_\infty$). If $0 \le j \le L$, then the length $T_j$ of the $j$-th recovery run is almost a geometric process: While the number of susceptible individuals stays at $S^{(j)}$, we are waiting for the next infection ('success'), which comes at each active step independently with probability

$$\mathbf{Pr}\left[I_{t+1} = I_t + 1 \mid I_{t+1} \neq I_t \text{ (active st) and } S_t = S^{(j)}\right] = \frac{\beta S^{(j)}}{\beta S^{(j)} + \gamma N} = p_j \,. \tag{10}$$

It is not exactly a geometric process, since we stop waiting for a success also when all remaining infected individuals happen to recover (no next infection). More precisely, for $0 \le j \le L$, $T_j = \min\left\{\widetilde{T}_j, I^{(j-1)} + 1\right\}$ where $\widetilde{T}_j + 1$ are independent geometric variables with the parameters $p_j$, and $I^{(j-1)} + 1$ is the number of infected individuals at the beginning of the $j$-th recovery run. (Set $I^{(-1)} = I_0 - 1$, to make $T_j$ work also for $j = 0$). We derive the expectation and concentration of $L$ by referring to the equation $I_0 + L - T(L) = 0$ and by finding $k$ such that $I_0 + k - \mathbf{E}\left[\widetilde{T}(k)\right] = O(1)$ and $k = \Theta(N)$ (11), where $\widetilde{T}(k) = \widetilde{T}_0 + \widetilde{T}_1 + \cdots + \widetilde{T}_k$. Let $k_r$ be the solution to (11). We will prove that $k_r \sim$ r.h.s. of (2). Before showing this, we first argue that $|L - k_r| = O(\sqrt{N}\log^3 N)$ with either (polynomially) high probability (w.h.p.) or with probability at least $1 - \exp(-\Omega(I_0))$, which ever is smaller. This implies that $\mathbf{E}[R_\infty] \sim \mathbf{E}[L] \sim k_r$ (since $k_r = \Theta(N)$ and $L = O(N)$) and $R_\infty$ is concentrated around its expectation.

While solving (11), we will observe that for $k \le k_r$ such that $k_r - k = o(N)$, $I_0 + k - \mathbf{E}\left[\widetilde{T}(k)\right] = \Theta(k_r - k)$. Based on this, we take $k' = k_r - \Theta(\sqrt{N}\log^3 N)$ such that $I_0 + k' - \mathbf{E}\left[\widetilde{T}(k')\right] = (1 + o(1))\sqrt{N}\log^3 N$. Informally, we are not looking at infection $k_r$, which might not even occur, but instead at an earlier infection $k' < k_r$, ensuring that at this point, while the process is dying out, there are still $\Theta(\sqrt{N}\log^3 N)$ infections left to clear. We distinguish between two cases based on $I_0$. First assume $I_0 \ge 2\sqrt{N}\log^3 N$. For $k$ increasing from 0 to $k'$, the function $k - \mathbf{E}\left[\widetilde{T}(k)\right]$ is bitonic (first increasing and then decreasing), so for each $0 \le k \le k'$, $I_0 + k - \mathbf{E}\left[\widetilde{T}(k)\right] \ge \sqrt{N}\log^3 N$. As $\widetilde{T}(k)$ is the sum of fewer than $N$ independent geometric random variables, each with the probability of success at least some positive constant, it can be shown that the probability that $\widetilde{T}(k)$ differs from its expectation by at least $\sqrt{N}\log^3 N$ is at least $e^{-\Omega(\log^2 N)}$ (for example, by using the Azuma–Hoeffding inequality, considering separately the case when the value of one of the geometric variables is greater than $\log^2 N$). Thus, by the union bound, with high probability (w.h.p.), for each $0 \le k \le k'$, $I_0 + k - \widetilde{T}(k) > 0$, and also $I_0 + k' - \widetilde{T}(k') \le 2\sqrt{N}\log^3 N$. This implies that, w.h.p. for each $0 \le k \le k'$, $T_k = \widetilde{T}(k)$, so, w.h.p. $L \ge k'$ and the number of active infections after the $k'$ recovery run is $I^{(k')} = I_0 + k' - T(k') = I_0 + k' - \widetilde{T}(k') = O(\sqrt{N}\log^3 N)$. The probability of infection at each active step after the $k'$ infections is a constant less than $1/2$ ($p_{k_r}$ is a constant less than $1/2$ and $p_{k'} = p_{k_r} + o(1)$) so the process finishes w.h.p. within additional $O(\sqrt{N}\log^3 N)$ active steps, that is, $L = k' + O(\sqrt{N}\log^3 N) = k_r + O(\sqrt{N}\log^3 N)$.

For $I_0 = \omega(1)$ but less than $2\sqrt{N}\log^3 N$, we reduce to the previous case by observing that with probability at least $1 - \exp -\Omega(I_0)$, the number of active infections increases to $2\sqrt{N}\log^3 N$, while the number of recoveries increases by $\Theta(\sqrt{N}\log^3 N)$. This can easily be done by using the proof idea of Section 3.

We now come back to solving (11) for $k$. The expectation of $\widetilde{T}_j$ is equal to $1/p_j - 1$, so the expectation of $\widetilde{T}(k)$ is

$$\mathbf{E}\left[\widetilde{T}(k)\right] = \sum_{j=0}^{k} \frac{\gamma}{\beta}\frac{N}{S_0 - j} = \frac{N\gamma}{\beta}\left(\sum_{j=S_0-k}^{S_0}\frac{1}{j}\right) = \frac{N\gamma}{\beta}\left(\log\left(\frac{S_0}{S_0-k}\right) + O\left(\frac{1}{S_0-k}\right)\right)$$

The r.h.s. above is equal to $I_0 + k$ when $\frac{I_0+k}{N}\frac{\beta}{\gamma} - O\left(\frac{1}{S_0-k}\right) = \log\left(\frac{S_0}{S_0-k}\right)$, yielding $1 - \frac{k}{S_0} = \exp\left(-\frac{I_0+k}{N}\frac{\beta}{\gamma} + O\left(\frac{1}{S_0-k}\right)\right)$. From this equation, putting $\kappa = k/N$, using the assumption that $S_0 = N - o(N)$, and considering $N$ increasing to infinity, we get $1 - \kappa = 1 - \frac{k}{N} = e^{-(\beta/\gamma)\kappa}\left(1 + O\left(\frac{I_0}{N}\right)\right) = e^{(\beta/\gamma)(1-\kappa)-\beta/\gamma}\left(1 + O\left(\frac{I_0}{N}\right)\right)$, so $-(\beta/\gamma)(1-\kappa)e^{-(\beta/\gamma)(1-\kappa)} = -(\beta/\gamma)e^{-(\beta/\gamma)}\left(1 + O\left(\frac{I_0}{N}\right)\right)$, giving a solution $\kappa_r = 1 + \frac{\gamma}{\beta}W\left(-(\beta/\gamma)e^{-(\beta/\gamma)}\right) + O\left(\frac{I_0}{N}\right)$. Thus $k_r = \kappa_r N \sim$ r.h.s. of (2).

# C APPENDIX: PROOF OF PROPOSITION 1

*Proof.* To show this result we will use an identity coupling. Hence, we assume that both processes activate the same individuals and make the same random decisions. It is easy to

see that the result holds if $T = T_\tau$ (both processes stopped for the same reason). Hence, to proof the lemma we have to estimate the probability that $T_\tau < T$.

In the following we will consider the first $\tau$ time steps of $P$ and $P_{\tau,*}$. We consider two cases: $\beta > \gamma + \varepsilon$ or $\beta < \gamma - \varepsilon$.

**Case $\beta > \gamma + \varepsilon$:** We consider a slightly different process $P'$ in which we always keep at least one infected individual (if there is only one infected individual we do not allow that individual to recover). For $P'$ we show that the probability of hitting $\sqrt{N}$ many infected individuals during the first $\tau$ time steps is at least $1 - 1/N$. Note that this event ($P'$ hitting $\sqrt{N}$ many infected individuals during the first $\tau$ steps) implies that process $P$ hits either $\sqrt{N}$ or 0 many infected individuals during the first $\tau$ time steps, so we will conclude that $\mathbf{Pr}[T_\tau = T] \geq 1 - 1/N$, or equivalently that $\mathbf{Pr}[T_\tau < T] \leq 1/N$.

It remains to show that the probability for $P'$ to hit $\sqrt{N}$ many infected individuals in the first $\tau$ time steps is at least $1 - 1/N$. Since we only consider the first $\tau$ time steps of $P'$ we know that $S_t \geq N - \tau$. Fix an active step $t' \leq \tau$. Define $\delta = \frac{\varepsilon}{4\beta}$. The probability that the number of infections is increased by one is

$$\begin{aligned}\frac{\frac{S_{t'}}{N} \cdot \beta \cdot \frac{I_{t'}}{N}}{\frac{S_{t'}}{N} \cdot \beta \cdot \frac{I_{t'}}{N} + \gamma \cdot \frac{I_{t'}}{N}} &= \frac{\beta}{\beta + \frac{N}{S_{t'}} \cdot \gamma} \geq \frac{\beta}{\beta + \frac{N}{N-\tau} \cdot \gamma} \\ &= \frac{\beta}{2\beta - \left(\beta - \frac{N}{N-\tau} \cdot \gamma\right)} \\ &> \frac{1}{2} + \frac{\beta - \gamma}{4\beta} = \frac{1}{2} + \delta,\end{aligned}$$

where the last inequality holds for sufficiently large $N$. Hence, in $\tau' = \sqrt{N} \log N$ many steps the expected number of infections is increased at least $\left(\frac{1}{2} + \delta\right) \sqrt{N} \log N$ many times. Similarly, since the probability that the number of infections is decreased by one in step $t'$ is at most $\frac{1}{2} - \delta$, in $\tau' = \sqrt{N} \log N$ many steps the expected number of decreases is at most $\left(\frac{1}{2} - \delta\right) \sqrt{N} \log N$. Using the Chernoff bounds from Lemma 1, together with the union bound, we can show that with a probability of $1 - 1/N$ the number of increases is at least $\left(\frac{1}{2} + \frac{\delta}{2}\right) \sqrt{N} \log N$ and the number of decreases is at most $\left(\frac{1}{2} - \frac{\delta}{2}\right) \sqrt{N} \log N$. Hence with probability at least $1 - 1/N$ we have $I_\tau \geq \sqrt{N}$.

**Case $\beta < \gamma - \varepsilon$:** This proof is identical to that of Case 1. The only difference is now that we show that it is very likely to have much more decreases such that $I_\tau = 0$.

□

**Proposition 4.** *Let $\mathcal{E}'$ be the event that $R_{T_\tau} \leq \sqrt{N} \log N/2$ and $I_{T_\tau} = \sqrt{N}$. Then,*

$$\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right] \approx \mathbf{E}\left[r_\infty \mid \mathcal{E}'\right]. \tag{12}$$

*Moreover,*

$$\mathbf{E}\left[r_\infty \mid \mathcal{E}_{ext}\right] = o(1). \tag{13}$$

*Proof of Proposition 4.* Note that $I_T^{(P)} = I_{T_\tau}^{(P_{\tau,*})}$ implies that $R_{T_\tau}^{(P_{\tau,*})} \leq \frac{\sqrt{N} \log N}{2}$. By Proposition 1, $\mathbf{Pr}\left[I_T^{(P)} = I_{T_\tau}^{(P_{\tau,*})}\right] \geq 1 - 1/N$. Thus,

$$\begin{aligned}\mathbf{Pr}\left[\overline{\mathcal{E}'} \mid \overline{\mathcal{E}_{ext}}\right] &= \frac{\mathbf{Pr}\left[\overline{\mathcal{E}'}, \overline{\mathcal{E}_{ext}}\right]}{\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right]} \\ &= \frac{\mathbf{Pr}\left[R_{T_\tau} > \sqrt{N} \log N/2,\ I_{T_\tau} = \sqrt{N}\right]}{\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right]} \\ &\leq \frac{\mathbf{Pr}\left[I_T^{(P)} \neq I_{T_\tau}^{(P_{\tau,*})}\right]}{\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right]} \leq \frac{1/N}{\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right]} = O\left(\frac{1}{N}\right),\end{aligned}$$

since we assume $\beta$ and $\gamma$ to be bounded by constants. From this, we get

$$\mathbf{Pr}\left[\mathcal{E}' \mid \overline{\mathcal{E}_{ext}}\right] \in [1 - O(1/N), 1].$$

Note that $\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}, \overline{\mathcal{E}'}\right] \leq 1$. Thus, asymptotically, we derive

$$\begin{aligned}\mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right] &= \mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}, \mathcal{E}'\right] \cdot \mathbf{Pr}\left[\mathcal{E}' \mid \overline{\mathcal{E}_{ext}}\right] \\ &\quad + \mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}, \overline{\mathcal{E}'}\right] \cdot \mathbf{Pr}\left[\overline{\mathcal{E}'} \mid \overline{\mathcal{E}_{ext}}\right] \\ &\approx \mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}, \mathcal{E}'\right] = \mathbf{E}\left[r_\infty \mid \mathcal{E}'\right].\end{aligned} \tag{14}$$

Using Similar arguments, one can show that $\mathbf{E}\left[r_\infty \mid \mathcal{E}_{ext}\right] = o(1)$.

□

# D APPENDIX: PROOF OF PROPOSITION 2

*Proof.* First we consider the case $S = N$ and show $\mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,*})} = \sqrt{N}\right] \leq \mathbf{Pr}\left[I_{T_\tau}^{(P_{\tau,N})} = \sqrt{N}\right]$. Via induction we show that for all $t \leq T_\tau$ we have $|I_{T_\tau}^{(P_{\tau,*})}| \leq |I_{T_\tau}^{(P_{\tau,N})}|$. The result follows directly form the induction. Since both processes have exactly one infected individual at $t = 0$ the base case certainly holds. Now we have to show that $|I_{t+1}^{(P_{\tau,*})}| \leq |I_{t+1}^{(P_{\tau,N})}|$. Note that, since $T_\tau = \sqrt{N} \log N$, both processes have susceptible individuals to turn into infected individuals. Hence, $\mathbf{Pr}\left[I_{t+1}^{(P_{\tau,*})} = I_t^{(P_{\tau,*})} + 1\right] = \frac{I_t^{(P_{\tau,*})}}{N} \cdot \beta \cdot \frac{S_t^{(P_{\tau,*})}}{N} \leq \frac{I_t^{(P_{\tau,N})}}{N} \cdot \beta = \mathbf{Pr}\left[I_{t+1}^{(P_{\tau,N})} = I_t^{(P_{\tau,N})} + 1\right]$. If $I_t^{(P_{\tau,*})} = I_t^{(P_{\tau,N})}$ then both processes have the same probability $\frac{I_t^{(P_{\tau,*})}}{N} \cdot \gamma$

to decrease the number of infections by one. If $I_t^{(P_{\tau,N})} < I_t^{(P_{\tau,N})}$ the probability for a decrease is slightly larger for $P_{\tau,N}$ but we still have $|I_{t+1}^{(P_{\tau,*})}| \leq |I_{t+1}^{(P_{\tau,N})}|$. In both cases we have $|I_{t+1}^{(P_{\tau,*})}| \leq |I_{t+1}^{(P_{\tau,N})}|$.

Now we are ready for the case $S = N - \tau$. Again, via induction we show that for all $t \leq T_\tau$ we have $|I_{T_\tau}^{(P_{\tau,N})}| \leq |I_{T_\tau}^{(P_{\tau,N})}|$. The base case trivially holds again. Since $\frac{S_t^{(P_{\tau,*})}}{N} \geq N - \tau$ we get $\mathbf{Pr}\left[I_{t+1}^{(P_{\tau,N-\tau})} = I_t^{(P_{\tau,N-\tau})} + 1\right] = \frac{I_t^{(P_{\tau,N-\tau})}}{N} \cdot \beta \cdot \frac{N-\tau}{N} \leq \frac{I_t^{(P_{\tau,*})}}{N} \cdot \beta \cdot \frac{S_t^{(P_{\tau,*})}}{N} = \mathbf{Pr}\left[I_{t+1}^{(P_{\tau,N})} = I_t^{(P_{\tau,N})} + 1\right]$. If $I_t^{(P_{\tau,N-\tau})} = I_t^{(P_{\tau,*})}$ then both processes have the same probability to decrease the number of infections by one. If $I_t^{(P_{\tau,N-\tau})} < I_t^{(P_{\tau,N})}$ the probability for a decrease is slightly larger for $P_{\tau,N}$, but we still have $|I_{t+1}^{(P_{\tau,*})}| \leq |I_{t+1}^{(P_{\tau,N})}|$. □

## E APPENDIX: MISSING PROOFS

**Proof of Theorem 2.** By law of total expectation, we can bound the term $\mathbf{E}[r_\infty]$ as follows

$$\mathbf{E}[r_\infty] = \mathbf{E}[r_\infty \mid \mathcal{E}_{ext}]\,\mathbf{Pr}[\mathcal{E}_{ext}] + \mathbf{E}\left[r_\infty \mid \overline{\mathcal{E}_{ext}}\right]\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right] \tag{15}$$

$$\approx \mathbf{E}[r_\infty \mid \mathcal{E}']\,\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right] \tag{16}$$

The bound follows by using Proposition 4 and plugging in the value for $\mathbf{E}[r_\infty \mid \mathcal{E}']$ (Theorem 6) and the value for $\mathbf{Pr}\left[\overline{\mathcal{E}_{ext}}\right]$ (Theorem 4). □

**Proof of Theorem 3.** The result follows from Theorem 6 and Proposition 4. □

## F APPENDIX: CONVEXITY OF $\mathfrak{R}_0$

See Fig. 4.

## G APPENDIX: AUXILIARY CLAIMS

**Theorem 7** (Chapter XIV.2, XIV.3 in Feller [1968]). *Let $p \in (0,1) \setminus \{1/2\}$ and $b, s \in \mathbb{N}$. Consider a discrete time Markov chain $(Z_t)_{t\geq 0}$ with state space $\Omega = [0, b]$ where*

- $Z_0 = s \in [0, b]$
- $\mathbf{Pr}[Z_t = i \mid Z_{t-1} = i - 1] = p$ *for* $i \in [1, b-1], t \geq 1$
- $\mathbf{Pr}[Z_t = i \mid Z_{t-1} = i + 1] = 1 - p$ *for* $i \in [1, b-1], t \geq 1$

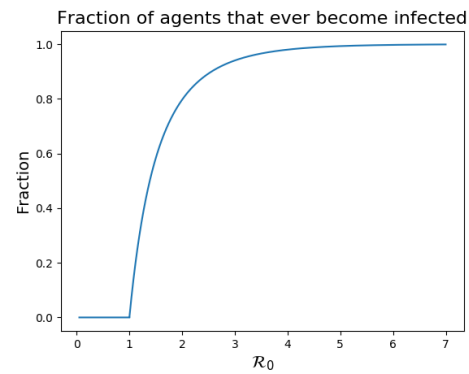



Figure 4: The plot shows $r_\infty$ for different values of $\mathfrak{R}_0 = \beta/\gamma$ and $s = 1$.

- $\mathbf{Pr}[Z_t = i \mid Z_{t-1} = i] = 1$ *for* $i \in \{0, b\}, t \geq 1$

*Let* $T = \min\{t \geq 0 \mid Z_t \in \{0, b\}\}$. *Then,*

$$\mathbf{Pr}[Z_T = b] = \frac{\left(\frac{1-p}{p}\right)^s - 1}{\left(\frac{1-p}{p}\right)^b - 1} \text{ and}$$

$$\mathbf{Pr}[Z_T = 0] = \frac{\left(\frac{1-p}{p}\right)^b - \left(\frac{1-p}{p}\right)^s}{\left(\frac{1-p}{p}\right)^b - 1}.$$

*Moreover,*

$$\mathbf{E}[T] = \frac{s}{1-2p} - \frac{b}{1-2p} \cdot \frac{1 - \left(\frac{1-p}{p}\right)^s}{1 - \left(\frac{1-p}{p}\right)^b}.$$

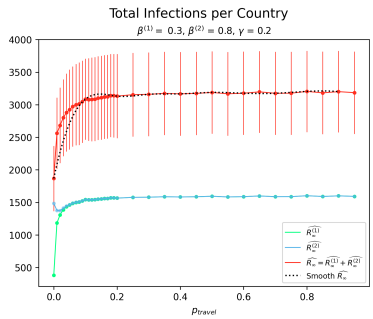
**Lemma 1** (Chernoff bounds). *Let $X_1 \ldots X_n$ be independent random variables taking values in $[0, 1]$. Let $X = \sum_{i=1}^n X_i$. Then for all $\lambda \geq 0$*

$$\mathbf{Pr}[X \geq \mathbf{E}[X] + \lambda] \leq e^{-\frac{2\lambda^2}{N}}.$$

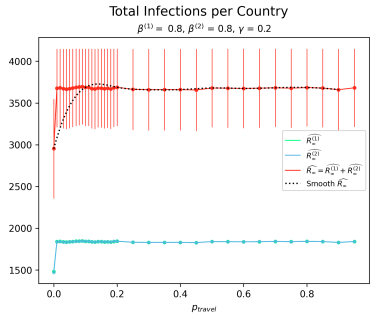
$$\mathbf{Pr}[X \geq \mathbf{E}[X] - \lambda] \leq e^{-\frac{2\lambda^2}{N}}.$$

## H APPENDIX: FURTHER SIMULATIONS PLOTS

**Simulation Environment:** or all the simulations, the language used is C++. Simulations were run on a DELL Latitude 7400 laptop, with Intel Core i7-8665U, vPro - 8th Gen CPU and 16GB RAM. We plot the mean value and the standard deviation.




(a) $\beta^{(1)}, \beta^{(2)} > \gamma$




(b) $\beta^{(1)} = \beta^{(2)} >> \gamma$

Figure 5: The total number of infections $\widehat{R_\infty}(p_{travel})$ appears to increase monotonically. Note that, in the plot below, $R(0) \neq R(p)\ for\ p > 0$ suggesting that $\mathbf{Pr}\left[\mathcal{E}_{ext}\right]$ might prevent the process to have the same outcome as if the two $\beta$s were replaced by their average.